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Learned pain behaviour

Pain is a symptom and does not necessarily indicate physical injury. While the relation between acute pain and tissue damage is close, patients with persistent pain who are referred to doctors often describe more pain than appears warranted from any pathological process that is present. These patients are usually described as suffering from psychogenic[1] or non-organic pain[2] and may be referred for psychiatric or psychological help. Some have clear evidence of psychiatric illness—in particular depression[3,4]—but in others pain may develop or persist independently of any mental illness. How does this occur?

When we experience pain we may respond by, for example, changing posture or taking analgesics. If this action leads to a reduction in pain the same response is likely to be repeated in similar circumstances; if it is not effective other manoeuvres may be tried. In the same way if expressions of pain produce sympathetic attention (or any other desired reaction) from someone important to the victim and close at hand this will encourage future complaints of pain in the presence of the same person. Complaints may also enable the victim to avoid other unpleasant activities, thus further indirectly rewarding the sick role. Behaviours that are compatible with being well are not rewarded and so tend to be extinguished; passive behaviours, such as watching television, may be encouraged.[5] Often an oversolicitous parent or spouse may be encouraging this development[6,7]—a syndrome termed operant or learned pain behaviour.[8]

Clearly this behaviour may occur in patients with physical injury; the concept is not synonymous with non-organic pain. Recognition of learned pain behaviour depends on paying attention to the relation between pain behaviours and their apparent consequences—not on eliciting a multitude of inappropriate organic signs or symptoms. Certainly the patient should be seen and examined in detail, but those closest to him should ideally also be seen and the interactions between them and the patient observed. The transaction between the patient and the doctor may not be representative.

With this caveat in mind, warning signs from the history include improbable descriptions of the pain—for example, whole leg pain[9]—using affective words like "sickening" and "blinding" to describe it,[10] progression of the severity and extent of the pain over time,[4] and multiple treatments.[11] The patient may evidence exaggerated facial expression of pain, abnormal posture, frequent grimacing and sighing, and rubbing of the affected parts.[12] Past experience and personality factors may be of value. Patients with previous devotion to the work ethic,[13] adoption of an adult role early in childhood,[14] those who have been able to receive attention and help during life only by complaining of pain,[15] and patients who have been brought up in a household with a chronically sick relative[16] may be particularly susceptible to learned pain behaviour. Engel describes these patients as masochistic[13]; Szasz calls them "les hommes douloureux."[17] Both call them pain prone. Such individuals score highly on questionnaires designed to measure illness behaviour, though these instruments cannot be diagnostic on their own.[18]

Physical examination may elicit non-organic physical signs. These have been best worked out in relation to low back pain; they include over-reaction to examination, superficial skin tenderness, distribution of sensory or motor abnormalities that are not dermatomic in distribution, and simulation and distraction tests.[2] Excessive guarding and bracing movements may also be found.[19]

The management of these patients varies considerably. Once all information is to hand a clear explanation should be given to the patient and his relatives of what physical causes, if any, are contributing to the pain. If treatment is envisaged directed at changing the pain behaviour, in most cases the patient should be referred to a psychologist or behaviourally oriented psychiatrist. Only the well motivated should be selected. Treatment consists of eliminating the rewards resulting from pain behaviour and substituting more active and constructive behaviours that are encouraged appropriately.[5,20,21] Those closest to the patient must be brought into the treatment. These patients are not easy to help, especially those who have had pain for many years. To attempt to change the habits of a lifetime—particularly if these remain advantageous to the patient and his family—may prove an unreasonable contract; the therapist is entitled to retire to the wings if no progress is possible.

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The kidney in myeloma

"The tube contains urine of very high specific gravity. When boiled it becomes slightly opaque. On the addition of nitric acid, it effervesces, assumes a reddish hue, and becomes quite clear; but as it cools, assumes the consistence and appearance which you see. Heat reliquefies it. What is it?"[1] So wrote Dr Thomas Watson in 1845 to Henry Bence Jones about the urine of Thomas Alexander McBean, drawing attention to one of the most characteristic abnormalities of multiple myeloma.

Over half of all patients with myeloma develop renal insufficiency and it is the second most common cause of death (after infection).[2,3] A raised blood urea concentration at presentation is the single most important pointer to a poor prognosis.[4]

The causes of renal impairment in myeloma are many, and any one patient is unlikely to have a single cause. Those factors most usually mentioned include infection, hypercalcaemia, hyperuricaemia, hyperviscosity, Bence Jones proteinuria, Fanconi's syndrome, plasma cell infiltration of the kidney, amyloidosis, and glomerulosclerosis.[5]

Renal failure is strongly associated with an excess of immunoglobulin light chains in the urine. Light chains are normally filtered by glomeruli and then reabsorbed and catabolised by cells in the proximal tubule—a physiological mechanism designed to deal with the small amounts of free light chain produced by normal people. The large amounts produced in myeloma, however, are toxic to renal tubular cells. In animals, injecting large amounts of light chain causes acute renal damage associated with the formation of casts and tubular atrophy,[6] and studies of slices of rat kidney incubated with light chains have shown inhibition of the adenosine triphosphatase dependent sodium pump[7] and of gluconeogenesis and metabolism of sodium iodohippurate in tubular cells.[8]

Yet despite this toxicity some patients excrete large quantities of free light chains for long periods without any damage to their renal tubules.[9] Apparently some light chains are more toxic than others. Two groups have shown that renal damage in myeloma correlates with the presence of urinary light chains with a high isoelectric point (pI).[10,11] Such light chains are more likely to precipitate in the acid urine of distal tubules to form casts; but attempts to alkalise the urine with oral bicarbonate in a large series of patients had no effect on survival.[12] This finding may merely show the difficulty of alkalising the urine.

The casts in the urine of patients with myeloma are characteristic, having a waxy, laminated structure surrounded by reactive, syncytial giant cells, with occasional renal cells embedded in the matrix.[13] Such casts usually indicate renal failure,[13] and it was once thought that they caused the damage in myeloma kidney by obstructing individual nephrons.[14] As many as a third of patients with myeloma, however, have no kidney casts,[5,15,16] and they are more likely to be the consequence of renal damage than its cause.

The sequence of events in myeloma kidney begins with insidious damage to the proximal tubular cells by filtered light chains. This is present in virtually all patients with urinary light chain concentrations of over one unit per litre whether or not glomerular function is impaired.[17] As a result tubular reabsorption of light chains is reduced, increasing their final concentration in the urine. Tubular damage may be compounded by other factors such as hypercalcaemia, hyperuricaemia, and nephrotoxic antibiotics. Against this background individual episodes of dehydration and infection lead to tubular atrophy.[5] As nephrons are lost each individual tubule carries an increasing load of light chain resulting in the formation of casts—possibly owing to the interaction of cationic light chains with the anionic Tamm-Horsfall mucoprotein.[6]

Patients who present with renal failure commonly do so after a recent precipitating event, usually infection or dehydration. Prompt treatment with rehydration, antibiotics, and regimens to lower the calcium and urate concentrations—together with short term dialysis if necessary—will often restore renal function.[18] The eventual outcome depends on whether or not the tumour responds to chemotherapy.

In patients who have urinary light chains but who are not yet in renal failure a fluid intake of three litres a day is likely to prevent deterioration of renal function.[12] The exception to this happy prognosis is renal amyloidosis, which causes glomerular lesions as well as interstitial damage. Although individual patients have responded to intensive chemotherapy,[19,20] in general even experimental treatments have been unsuccessful in this condition.[21] Nevertheless, though systemic amyloidosis in myeloma is usually rapidly lethal, its progression is sometimes slow; it is well worth persevering with treating the myeloma and the renal failure with supportive measures short of long term dialysis.

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